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**Search Results -**

Term	Documents
B7-1.USPT.	145
B7-1S	0
B7.USPT.	6721
B7S	0
ANTIBOD\$	0
ANTIBOD.USPT.	212
ANTIBODAY.USPT.	1
ANTIBODEES.USPT.	1
ANTIBODEIS.USPT.	1
ANTIBODES.USPT.	43
.....	
TREAT\$(TREATMENT/ULTRAFILTRATION).USPT.	pickup term
((('B7-1' OR 'B7') SAME ANTIBOD\$ AND (INHIBIT\$ OR TREAT\$ OR SUPPRESS\$ OR THERAP\$) SAME (PSORIASIS) ).USPT.	44

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EPO Abstracts Database

Derwent World Patents Index

Database:

IBM Technical Disclosure Bulletins

Refine Search:

('b7-1' or 'b7') same antibod\$ and  
(inhibit\$ or treat\$ or suppress\$ or  
therap\$) same (psoriasis)

Clear

**Search History**

**Today's Date: 2/25/2001**

<u>DB Name</u>	<u>Query</u>	<u>Hit Count</u>	<u>Set Name</u>
USPT	('b7-1' or 'b7') same antibod\$ and (inhibit\$ or treat\$ or suppress\$ or therap\$) same (psoriasis)	44	<u>L1</u>

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? set hi ;set hi
HIGHLIGHT set on as ''
HIGHLIGHT set on as ''
? begin 5,73,155,399
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      $0.00      0.071 DialUnits File410
$0.00 Estimated cost File410
$0.01 TELNET
$0.01 Estimated cost this search
$0.33 Estimated total session cost      0.162 DialUnits

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SYSTEM:OS - DIALOG OneSearch
  File 5:Biosis Previews(R) 1969-2003/Aug W1
    (c) 2003 BIOSIS
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*File 155: Medline has been reloaded and accession numbers have
changed. Please see HELP NEWS 155.
  File 399:CA SEARCH(R) 1967-2003/UD=13906
    (c) 2003 American Chemical Society
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Set Items Description
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? e au=anderson darrell ?

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Ref	Items	Index-term
E1	1	AU=ANDERSON DARREL R
E2	7	AU=ANDERSON DARRELL
E3	0	*AU=ANDERSON DARRELL ?
E4	4	AU=ANDERSON DARRELL E
E5	7	AU=ANDERSON DARRELL R
E6	1	AU=ANDERSON DARWIN
E7	8	AU=ANDERSON DARWIN W
E8	1	AU=ANDERSON DARYLL
E9	5	AU=ANDERSON DAVE
E10	82	AU=ANDERSON DAVID
E11	31	AU=ANDERSON DAVID A
E12	1	AU=ANDERSON DAVID ANDREW

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Enter P or PAGE for more

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? s e1-e7
      1 AU=ANDERSON DARREL R
      7 AU=ANDERSON DARRELL
      0 AU=ANDERSON DARRELL ?
      4 AU=ANDERSON DARRELL E
      7 AU=ANDERSON DARRELL R
      1 AU=ANDERSON DARWIN
      8 AU=ANDERSON DARWIN W
S1      28 E1-E7

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? rd s1
...completed examining records
      S2      27 RD S1 (unique items)
? s s2 and (b7? or cd80 or cd86)
      27 S2
      22132 B7?
      9225 CD80
      7523 CD86

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S3 3 S2 AND (B7? OR CD80 OR CD86)  
? t s3/3/all

3/3/1 (Item 1 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2003 BIOSIS. All rts. reserv.

13591346 BIOSIS NO.: 200200220167  
Therapeutic activity of IDEC-114 (anti-CD80) and rituximab  
(Rituxan(R)) in B-cell lymphoma.  
AUTHOR: Hariharan Kandasamy(a); **Anderson Darrell**(a); Leigh Bryan(a);  
Berquist Lisa G(a); Murphy Tracey(a); Leonard John E(a); Braslawsky Gary  
R(a); Hanna Nabil(a)  
AUTHOR ADDRESS: (a)IDEC Pharmaceuticals Corp., San Diego, CA\*\*USA  
JOURNAL: Blood 98 (11 Part 1):p608a November 16, 2001  
MEDIUM: print  
CONFERENCE/MEETING: 43rd Annual Meeting of the American Society of  
Hematology, Part 1 Orlando, Florida, USA December 07-11, 2001  
ISSN: 0006-4971  
RECORD TYPE: Abstract  
LANGUAGE: English

3/3/2 (Item 2 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2003 BIOSIS. All rts. reserv.

12995073 BIOSIS NO.: 200100202222  
Human B7.1-specific primatized antibodies and transfectomas  
expressing said antibodies.  
AUTHOR: **Anderson Darrell** R(a); Brams Peter; Hanna Nabil; Shestowsky  
William S; Heard Cheryl  
AUTHOR ADDRESS: (a)Escondido, CA\*\*USA  
JOURNAL: Official Gazette of the United States Patent and Trademark Office  
Patents 1238 (1):pNo Pagination Sep. 5, 2000  
MEDIUM: e-file  
ISSN: 0098-1133  
DOCUMENT TYPE: Patent  
RECORD TYPE: Abstract  
LANGUAGE: English

3/3/3 (Item 3 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2003 BIOSIS. All rts. reserv.

11995118 BIOSIS NO.: 199900275637  
In vitro immunological and functional properties of primate/human chimeric  
monoclonal (PRIMATIZED(R)) antibodies to human CD80.  
AUTHOR: Shestowsky William(a); Brams Peter(a); Pan Li-Zhen(a); Nguyen  
Mai-Lan(a); Chambers-Slater Karen(a); Franco Luis(a); Hanna Nabil(a);  
**Anderson Darrell**(a)  
AUTHOR ADDRESS: (a)IDEC Pharmaceuticals Corporation, 11011 Torreyana Road,  
San Diego, CA, 92121\*\*USA  
JOURNAL: FASEB Journal 13 (5 PART 2):pA953 March 15, 1999  
CONFERENCE/MEETING: Annual Meeting of the Professional Research Scientists  
on Experimental Biology 99 Washington, D.C., USA April 17-21, 1999  
SPONSOR: Federation of American Societies for Experimental Biology  
ISSN: 0892-6638  
RECORD TYPE: Citation  
LANGUAGE: English  
? s (b7? or cd80 or cd86) and (16C10 or 7C10 or 20C9 or 7B6)  
22132 B7?  
9225 CD80

7523 CD86  
1 16C10  
32 7C10  
16 20C9  
39 7B6

S4 7 (B7? OR CD80 OR CD86) AND (16C10 OR 7C10 OR 20C9 OR 7B6)  
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S5 3 RD S4 (unique items)  
? t s5/7/all

5/7/1 (Item 1 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2003 BIOSIS. All rts. reserv.

12995073 BIOSIS NO.: 200100202222  
Human B7.1-specific primatized antibodies and transfectomas  
expressing said antibodies.  
AUTHOR: Anderson Darrell R(a); Brams Peter; Hanna Nabil; Shestowsky William  
S; Heard Cheryl  
AUTHOR ADDRESS: (a)Escondido, CA\*\*USA  
JOURNAL: Official Gazette of the United States Patent and Trademark Office  
Patents 1238 (1):pNo Pagination Sep. 5, 2000  
MEDIUM: e-file  
ISSN: 0098-1133  
DOCUMENT TYPE: Patent  
RECORD TYPE: Abstract  
LANGUAGE: English

ABSTRACT: The present invention relates to the identification of macaque  
antibodies to human B7.1 and B7.2 by screening of phage  
display libraries or monkey heterohybridomas obtained using B lymphocytes  
from B7.1 and/or B7.2 immunized monkeys. More specifically,  
the invention provides four monkey monoclonal antibodies 7B6,  
16C10, 7C10 and 20C9 which inhibit the B7:CD28  
pathway and thereby function as effective immunosuppressants. The  
invention further provides the complete DNA and amino acid sequences of  
the light and heavy chain of three primatized antibodies derived from  
those monkey monoclonal antibodies which bind B7.1 and possibly  
B7.2, primatized 7C10, primatized 7B6 and primatized  
16C10. These primatized and monkey antibodies may be used as  
specific immunosuppressants, e.g., for the treatment of autoimmune  
diseases and to prevent organ transplant rejection.

5/7/2 (Item 2 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2003 BIOSIS. All rts. reserv.

09114917 BIOSIS NO.: 199497123287  
Induction of B cell costimulatory function by recombinant murine CD40  
ligand.  
AUTHOR: Kennedy Mary K(a); Mohler Kendall M; Shanebeck Kurt D; Baum Peter R  
; Picha Kathleen S; Otten-Evans Carol A; Janeway Charles A Jr; Grabstein  
Kenneth H  
AUTHOR ADDRESS: (a)Dep. Immunobiol., Immunex Res. Dev. Corp., 51 University  
St., Seattle, WA 98101\*\*USA  
JOURNAL: European Journal of Immunology 24 (1):p116-123 1994  
ISSN: 0014-2980  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: English

ABSTRACT: T cell-dependent regulation of B cell growth and differentiation

involves an interaction between CD40, a B cell surface molecule, and the CD40 ligand (CD40L) which is expressed on activated CD4+ T cells. In the current study, we show that recombinant membrane-bound murine CD40L induces B cells to express costimulatory function for the proliferation of CD4+ T cells. CD40L- or lipopolysaccharide (LPS)-activated, but not control-cultured B cells were strong costimulators of anti-CD3 or alloantigen-dependent T-cell responses. The molecular interactions responsible for the increased costimulatory functions were examined by analyzing the activated B cells for changes in the expression of two costimulatory molecules, B7 and heat-stable antigen (HSA), as well as by the use of antagonists of B7 and HSA (CTLA4.Fc and 20C9, respectively). The expression of both B7 and HSA was enhanced on B cells activated with LPS. As observed in previous studies, the costimulatory activity of the LPS-activated B cells was dependent on both B7 and HSA and was completely inhibited in the presence of a combination of CTLA4.Fc and 20C9. In contrast, activation of B cells with CD40L induced the expression of B7 but did not enhance the expression of HSA. In addition the costimulatory activity of the CD40L-activated B cells was partially, but not completely, inhibited by the combination of CTLA4.Fc and 20C9. These results demonstrate that CD40L regulates costimulatory function of B cells in part by inducing the expression of B7 and suggest that CD40L-activated B cells express an additional costimulatory activity that is not associated with LPS-activated B cells.

5/7/3 (Item 3 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2003 BIOSIS. All rts. reserv.

08738923 BIOSIS NO.: 199395028274  
Co-stimulation of murine CD4 T cell growth: Cooperation between B7 and heat-stable antigen.  
AUTHOR: Liu Yang(a); Jones Bryan; Brady William; Janeway Charles A Jr; Linley Peter S  
AUTHOR ADDRESS: (a)Div. Immunol., Dep. Pathol., New York Univ. Med. Cent., 550 First Ave., New York, N.Y. 10016\*\*USA  
JOURNAL: European Journal of Immunology 22 (11):p2855-2860 1992  
ISSN: 0014-2980  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: English

ABSTRACT: The B cell activation antigen B7/BB1 has been shown to co-stimulate growth of human T cells by binding the T cell molecule CD28. In mice, the heat-stable antigen (HSA) has also been shown to act as a co-stimulator for T cell growth. In this study, we have evaluated the contributions of B7 and HSA to the co-stimulatory activity of antigen-presenting cells (APC). Mouse B7 provides co-stimulatory activity for murine CD4 T cells in anti-CD3-induced proliferation. Human CTLA4Ig, a chimeric molecule comprising the extracellular region of CTLA-4 fused to an immunoglobulin C-gamma fragment, binds to murine B7. We, therefore, use human CTLA4Ig and the hamster anti-HSA monoclonal antibody 20C9 to analyze the relative contributions of B7 and HSA to the co-stimulatory activity of murine spleen APC. Our data reveal that both murine B7 and HSA are expressed by dendritic cells and by low-density spleen B cells. Either CTLA4Ig alone or anti-HSA alone inhibited CD4 T cell proliferation to anti-CD3 by gt 90%, while CTLA4Ig and anti-HSA together were far more efficient in inhibiting clonal expansion of CD4 T cells. These results demonstrate that functionally defined co-stimulation involves at least B7 and HSA and suggest that signals delivered by B7 and HSA synergize in promoting T cell growth.

? s (b7? or cd80 or cd86) and (psoriasis)

22132 B7?

9225 CD80

7523 CD86

55704 PSORIASIS

S6 166 (B7? OR CD80 OR CD86) AND (PSORIASIS)

? rd s6

...examined 50 records (50)

...examined 50 records (100)

...examined 50 records (150)

...completed examining records

S7 108 RD S6 (unique items)

?

7/7/16 (Item 16 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
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11985665 BIOSIS NO.: 199900266184

CTLA4Ig-mediated blockade of T-cell costimulation in patients with  
**psoriasis vulgaris**.

AUTHOR: Abrams Judith R(a); Lebwohl Mark G; Guzzo Cynthia A; Jegasothy  
Brian V; Goldfarb Michael T; Goffe Bernard S; Menter Alan; Lowe Nicholas  
J; Krueger Gerald; Brown Michael J; Weiner Russell S; Birkhofer Martin J;  
Warner Garvin L; Berry Karen K; Linsley Peter S; Krueger James G; Ochs  
Hans D; Kelley Susan L; Kang Sewon

AUTHOR ADDRESS: (a)Bristol-Myers Squibb Pharmaceutical Research Institute,  
5 Research Parkway, Wallingford, CT, 064\*\*USA

JOURNAL: Journal of Clinical Investigation 103 (9):p1243-1252 May, 1999

ISSN: 0021-9738

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

SUMMARY LANGUAGE: English

ABSTRACT: Engagement of the B7 family of molecules on  
antigen-presenting cells with their T cell-associated ligands, CD28 and  
CD152 (cytotoxic T lymphocyte-associated antigen-4 (CTLA-4)), provides a  
pivotal costimulatory signal in T-cell activation. We investigated the  
role of the CD28/CD152 pathway in **psoriasis** in a 26-week, phase I,  
open-label dose-escalation study. The importance of this pathway in the  
generation of humoral immune responses to T cell-dependent neoantigens,  
bacteriophage phiX174 and keyhole limpet hemocyanin, was also evaluated.  
Forty-three patients with stable **psoriasis vulgaris** received 4  
infusions of the soluble chimeric protein CTLA4Ig (BMS-188667). Forty-six  
percent of all study patients achieved a 50% or greater sustained  
improvement in clinical disease activity, with progressively greater  
effects observed in the highest-dosing cohorts. Improvement in these  
patients was associated with quantitative reduction in epidermal  
hyperplasia, which correlated with quantitative reduction in  
skin-infiltrating T cells. No markedly increased rate of intralesional  
T-cell apoptosis was identified, suggesting that the decreased number of  
lesional T cells was probably likely attributable to an inhibition of  
T-cell proliferation, T-cell recruitment, and/or apoptosis of  
antigen-specific T cells at extralesional sites. Altered antibody  
responses to T cell-dependent neoantigens were observed, but immunologic  
tolerance to these antigens was not demonstrated. This study illustrates  
the importance of the CD28/CD152 pathway in the pathogenesis of  
**psoriasis** and suggests a potential therapeutic use for this novel  
immunomodulatory approach in an array of T cell-mediated diseases.



7/7/13 (Item 13 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
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12465656 BIOSIS NO.: 200000219158

Results of a single-dose, dose-escalating trial of an anti-B7.1  
monoclonal antibody (IDEC-114) in patients with **psoriasis**.

AUTHOR: Gottlieb A(a); Abdulghani A; Totoritis M; Lizambri R; Shuey S;  
Romano P; Oh C; Chaudhari U; Lebwohl M

AUTHOR ADDRESS: (a)UMDNJ-RWJMS, New Brunswick, NJ\*\*USA

JOURNAL: Journal of Investigative Dermatology 114 (4):p840 April, 2000

CONFERENCE/MEETING: 61st Annual Meeting of the Society for Investigative  
Dermatology. Chicago, Illinois, USA May 10-14, 2000

ISSN: 0022-202X

RECORD TYPE: Citation

LANGUAGE: English

SUMMARY LANGUAGE: English

12552653 BIOSIS NO.: 200000306155

The role of co-stimulation in airway inflammation.

AUTHOR: Djukanovic R

AUTHOR ADDRESS: (a)University of Medicine, Southampton General Hospital,  
Tremona Road, Level D, Centre Block, Southampton, SO16 6YD\*\*UK

JOURNAL: Clinical and Experimental Allergy 30 (Supplement 1):p46-50 June,  
2000

MEDIUM: print

ISSN: 0954-7894

RECORD TYPE: Abstract

LANGUAGE: English

SUMMARY LANGUAGE: English

**ABSTRACT:** There is considerable evidence to support an important role for co-stimulatory molecules in regulating the proliferation and activation of T cells in the immune response. Of particular relevance is the interaction between CD28 on T cells and B7 expressed on the surface of antigen presenting cells (APCs). CTLA-4, another molecule present on activated T cells may downregulate T cell activity, but its role remains uncertain. CTLA4-Ig, a fusion protein consisting of the extracellular domain of CTLA4 and the Fc portion of human immunoglobulin G1 (IgG1), has been useful for studying the role of CD28/B7 interactions in immune responses. A number of studies have shown that CTLA4-Ig can switch off T cell activation. In an ovalbumin sensitive murine model of asthma, CTLA4-Ig treatment suppressed the response to inhaled allergen (increased airway hyperresponsiveness (AHR), IgE production, recruitment of eosinophils into the lungs, production of IL-4, IL-5, and IL-10 and increased IFNgamma production from CD3-TCR-activated T cells). Anti B7-2 treatment has similar effects suggesting that interaction of B7-2 with CD28 is important in the development of a Th-2 type inflammatory response in mice. Recent observations have been of relevance to human allergic disease. In vitro studies have shown that CTLA4-Ig or anti-B7-2 antibody can inhibit allergen-induced proliferation and cytokine production by peripheral blood mononuclear cells from atopic subjects. The role of co-stimulation has been studied in a human bronchial explant model of asthma. CTLA4-Ig fusion protein effectively blocked allergen-induced production of IL-5 and IL-13 in bronchial explants from atopic asthmatics. These studies confirm the requirement for interaction between co-stimulatory molecules in cytokine production and allergic inflammation, and point to the CD28-B7 pathway as being important to the allergen-induced inflammation in asthma. Studies of organ transplantation in primates suggest that CTLA4-Ig is extremely effective in preventing organ rejection. While phase 1 clinical trials have shown CTLA-4-Ig treatment of patients with psoriasis vulgaris to be well tolerated and to result in clinical improvement, its role in asthma management merits further investigation.

12666545 BIOSIS NO.: 200000420047

Blockade of T lymphocyte costimulation with cytotoxic T

lymphocyte-associated antigen 4-immunoglobulin (CTLA4Ig) reverses the cellular pathology of psoriatic plaques, including the activation of keratinocytes, dendritic cells, and endothelial cells.

AUTHOR: Abrams Judith R(a); Kelley Susan L; Hayes Elizabeth; Kikuchi Toyoko ; Brown Michael J; Kang Sewon; Lebwohl Mark G; Guzzo Cynthia A; Jegasothy Brian V; Linsley Peter S; Krueger James G

AUTHOR ADDRESS: (a)Novartis Pharmaceuticals Corporation, 59 Route 10, Bldg. 122, Rm. S320, East Hanover, NJ, 07936-1080\*\*USA

JOURNAL: Journal of Experimental Medicine 192 (5):p681-693 September 4, 2000

MEDIUM: print

ISSN: 0022-1007

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

SUMMARY LANGUAGE: English

ABSTRACT: Efficient T cell activation is dependent on the intimate contact between antigen-presenting cells (APCs) and T cells. The engagement of the B7 family of molecules on APCs with CD28 and CD152 (cytotoxic T lymphocyte-associated antigen 4 (CTLA-4)) receptors on T cells delivers costimulatory signal(s) important in T cell activation. We investigated the dependence of pathologic cellular activation in psoriatic plaques on B7-mediated T cell costimulation. Patients with **psoriasis** vulgaris received four intravenous infusions of the soluble chimeric protein CTLA4Ig (BMS-188667) in a 26-wk, phase I, open label dose escalation study. Clinical improvement was associated with reduced cellular activation of lesional T cells, keratinocytes, dendritic cells (DCs), and vascular endothelium. Expression of CD40, CD54, and major histocompatibility complex (MHC) class II HLA-DR antigens by lesional keratinocytes was markedly reduced in serial biopsy specimens. Concurrent reductions in B7-1 (CD80), B7-2 (CD86), CD40, MHC class II, CD83, DC-lysosomal-associated membrane glycoprotein (DC-LAMP), and CD11c expression were detected on lesional DCs, which also decreased in number within lesional biopsies. Skin explant experiments suggested that these alterations in activated or mature DCs were not the result of direct toxicity of CTLA4Ig for DCs. Decreased lesional vascular ectasia and tortuosity were also observed and were accompanied by reduced presence of E-selectin, P-selectin, and CD54 on vascular endothelium. This study highlights the critical and proximal role of T cell activation through the B7-CD28/CD152 costimulatory pathway in maintaining the pathology of **psoriasis**, including the newly recognized accumulation of mature DCs in the epidermis.

7/7/5 (Item 5 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
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13267288 BIOSIS NO.: 200100474437

**CD80/86 antisense oligonucleotide therapy improves psoriasis:**

Study in the SCID mouse model.

AUTHOR: Raychaudhuri S(a); Bennett C; Karras J; Mehta R; Chatterjee A(a);  
Farber E(a)

AUTHOR ADDRESS: (a)Psoriasis Research Institute, Palo Alto, CA\*\*USA

JOURNAL: Journal of Investigative Dermatology 117 (2):p443 August, 2001

MEDIUM: print

CONFERENCE/MEETING: 62nd Annual Meeting of the Society for Investigative  
Dermatology Washington, DC, USA May 09-12, 2001

ISSN: 0022-202X

RECORD TYPE: Citation

LANGUAGE: English

SUMMARY LANGUAGE: English

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13877222 BIOSIS NO.: 200200506043

Differentiation of monocyte-derived dendritic cells in patients with **psoriasis**.

AUTHOR: Zhu Kejian(a); Zhou Weifang(a); Lao Limin(a); Zheng Min(a)

AUTHOR ADDRESS: (a)Department of Dermatology, Second Affiliated Hospital, Zhejiang University School of Medicine, Hangzhou, 310009\*\*China

JOURNAL: Zhonghua Pifuke Zazhi 35 (2):p88-90 April, 2002

MEDIUM: print

ISSN: 0412-4030

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: Chinese; Non-English

ABSTRACT: Objective To investigate the differentiation of dendritic cells from monocytes of the peripheral blood in patients with **psoriasis**. Methods Flow cytometry was used to analyze the phenotype of monocyte-derived dendritic cells (MoDC). The capacity of MoDC to stimulate the proliferation of T lymphocytes was evaluated by allogeneic mixed lymphocyte reaction. Results Monocytes in the peripheral blood of patients with **psoriasis** could differentiate into dendritic cells. Expression of CD40, CD80, CD86 and HLA-DR by MoDC was significantly increased in patients with **psoriasis** compared with that in normal controls ( $P < 0.01$ ). The capacity of MoDC to stimulate T lymphocytes was enhanced significantly in patients' group as well ( $P < 0.01$ ). Conclusions The differentiation of monocytes into dendritic cells is upregulated in **psoriasis**, and these cells have potent antigen presenting capacities.